PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Punctiform Calcareous Bodies (so-called Calcified Glomeruli) of the Kidney Cortex.—BAUM (*Virchow's Archiv*, 1900, clxii., 85) has recently re-investigated, under the direction of Orth, the deposit of lime in the kidney.

Macroscopically the lime appears as small white lines and dots. Authorities generally agree that the lines are to be referred to its deposit in the tubules and in the interstitial tissue. Its situation, however, when it appears as points or dots has been a matter of dispute. Some have claimed that it was in the glomeruli, others that it was in the capsular space. Orth has recently expressed the opinion that it was deposited chiefly in the colloid contents of dilated tubules.

The writer investigated some twenty kidneys in which yellowish-white specks were more or less numerous in the cortex. Serial sections showed on microscopical examination two kinds of cysts present, although there was no evidence of chronic interstitial changes. The larger cysts were generally irregularly shaped, and their walls were lined in places with tall epithelium. The contents of the cysts were colloid. They were found both in the cortex and occasionally in the pyramids. The other cysts were round, about the size of a glomerulus, and confined to the cortex. The lining epithelium when present was low in type like that covering the capsular space of a glomerulus. Occasionally one or two loops suggesting a portion of a glomerulus projected from the wall. Evidently these cysts represent capsular spaces in which the glomeruli had not developed, and were to be regarded as congenital in origin.

It was found that the lime was deposited chiefly within these cysts in the colloid material filling them. It occurred as small granules and as concentrically layered masses. When fused together these masses formed a spherical ball. Only occasionally was calcification found to have taken place in a sclerosed glomerulus.

The Histology of Chronic Fibrous Pneumonia.—VOGEL (*Ziegler's Beiträge*, 1900, vol. xxviii., p. 179), working in Marchand's laboratory, studied ten cases of indurative pneumonia. Eight of the cases showed the organization of a pre-existing fibrinous intra-alveolar exudate. The two other cases showed a bronchial or a peribronchial inflammatory process which led to cellular infiltration and hyperplasia of the connective tissue of the lung. The end result of both processes is the same, namely, the replacement of the lung parenchyma by a more or less compact mass of connective tissue. In the first group of cases the newly formed connective tissue plugs contained in the alveoli enlarge and finally unite with the alveolar walls. In the second group the alveolar walls thicken and obliterate the alveoli.

The origin and development of the intra-alveolar connective tissue was investigated. When in acute pneumonia resolution does not occur the plugs of fibrin become retracted from the alveolar walls and clear spaces are formed in the periphery of the alveoli. In the first stage of the organization of the exudate, spindle-cells are found on the surface and advancing into the interior of the fibrinous plugs, and spindle cells are also seen extending along the threads of fibrin which pass through Cohn's pores. Gradually, connective tissue replaces the fibrinous masses. The connective tissue fibrillæ form a loose network at first, which contains in its meshes many plasma cells. Later the connective tissue becomes more compact. The bands which pass through Cohn's pores and the strands which unite the plugs to the alveolar wall become thicker until wall and contents become blended into one mass of fibrous tissue. In one case the new connective tissue contained delicate young elastic fibres.

Cohn thinks that the connective tissue arises from the interlobular and subpleural tissue, because he found the most numerous and the most developed connective tissue plugs in the neighboring alveoli. Ribbert maintains that the connective tissue begins to form in the smallest bronchi and bronchioles, and grows peripherally into the alveoli. Vogel opposes both these views. His observations lead him to believe, with von Kahlden and Borrmann, that the organization takes place from the alveolar wall. Not all the fine threads which suspend the connective tissue plugs in the centre of the alveoli pass to other plugs through the pores in the alveolar wall. Some are united to the wall; these he regards as primary outgrowths from the wall. Their delicate structure and the fact that they are found in the first stage of organization favor this view.

Vogel concludes that organization proceeds (1) from the alveolar wall into the fibrinous plugs; (2) from one fibrinous plug to another by the growth of connective tissue through Cohn's pores.—J. H. P.

Histological Changes Produced by Freezing.—RISCHPLER (*Ziegler's Beiträge*, 1900, vol. xxviii., p. 541) reports at considerable length the changes produced in animal tissues by freezing with ether spray. In his experiments he utilized the ear and the thigh of the rabbit and the tail of the mouse, keeping each part in a frozen condition for three minutes. Thirteen animals were used in each series and the tissues examined histologically at periods from twenty minutes to eight days after the freezing.

Rischpler found that all tissues examined (epidermis, muscle, fibrous tissue, tendon, cartilage, bone, nerves, and bloodvessels), with the exception of elastic fibres, show changes under the influence of a lower temperature. The extent of the change depends on the degree of cooling and the resistance of the tissue, the latter varying with size, structure, position, and nutrition. The mildest form of change is the appearance of vacuoles and fine granules in the protoplasm. The next grade is characterized by a shrinkage of cell and nucleus, the latter assuming bizarre shapes. Then comes loss of staining reaction in the protoplasm, and finally the destruction of the nucleus and disintegration of the cell.

These changes are an expression of cell death, and in those places where they are very frequent lead later to tissue necrosis. They are caused by the direct action of the cold on the tissue, and have no relation to thrombosed bloodvessels occasionally met, but the thrombi are due to these same changes affecting the bloodvessels. The injury is produced by the cold acting to abstract water from the water-containing components of the cell, and this water is converted into ice either within or without the cell. If this happens within the cell we find after thawing that the cell is made up of concentrated water—poor protoplasm, and of drops of water (in a stained section such a cell appears as a vacuolated cell); if without the cell, the cell and nucleus are found to be shrunken and distorted, while intercellular structures—*e. g.*, protoplasmic bridges of the epidermis—are destroyed or distorted. Beside necrosis inflammatory changes follow as a result of the tissue injury.

Exudation with fibrin formation appears in twenty minutes, and peripheral position of the leucocytes in bloodvessels and their emigration are noted in three-fourths of an hour after freezing. The cells slightly affected regain their normal appearance; those more seriously altered die and are replaced by regeneration from intact cells. The first signs of regeneration appear after six hours in the epidermis in the form of cells with large, constricted nuclei and multinuclear cells. These giant-cells are found also in the endothelium of arteries after twenty-four hours and in cartilage after three days. They are formed most probably by amitosis. After twenty-four hours mitotic figures are found in nearly all of the tissues. In muscle, regeneration begins in twenty-four hours, and many mitoses are found after three days. Regeneration progresses rapidly.—H. A. C.

The Presence of Fat in Pathological Tissues.—SATA (*Ziegler's Beiträge*, 1900, vol. xxviii., p. 461) gives the results of the examination of a number of pathological specimens with respect to the presence of fat. He finds that Sudan III. gives better results than the other fat-staining reagents. He fixes his tissues in formol and cuts sections with freezing microtome. The sections are dehydrated quickly in alcohol, stained with several changes of a saturated solution of Sudan III. in 95 per cent. alcohol, and mounted in glycerin. Sata insists on the necessity of studying both thin and thick sections. In struma, in the new-formed cells of inflammatory proliferation, in myoma, and in other pathological lesions he commonly finds fat present. In foci of necrosis there is usually a zone of fat droplets about the necrotic tissue. Their presence in the necrotic material is unusual, and when found their arrangement speaks for a previous existence in living cells. The same facts hold true for tubercular caseation, and as here the fat is absent in the early stages he thinks fatty degeneration has no causal relation to caseation.

In tumors in which no necrosis has taken place he very frequently finds great numbers of fat droplets, both in tumor and bordering stroma cells. The presence of fat seems to bear no relation to the age and nutrition of the cell. Connective tissue that has undergone a hyaline degeneration stains diffusely with Sudan III. In sarcoma cells fat droplets are not very common.

Sata thinks that the facts made out hardly support the view that the fat in these processes always arises through injury to the cell life in the sense of a fatty degeneration. He suggests that possibly in those cases in which necrosis is present the proteid has become so greatly changed that it can be readily broken down by the bordering cells, with the formation of fat. However, he does not deny that the living cell may be directly changed by chemical substances present in necrobiosis.—H. A. C.

The Occurrence of Lycopodium Spores within a Cancer of the Skin.—De-Meser (*Virchow's Archiv*, 1901, vol. clxiii., p. 111) examined a typical epidermoid carcinoma from the arm of an old man, and saw in the tumor peculiar three-cornered bodies which proved to be lycopodium spores. Inquiry showed that the man had been in the habit of dusting the malignant ulcer with lycopodium powder.

The spores usually were found lying in the connective tissue stroma, either free in clefts between connective tissue cells or enclosed in the protoplasm of giant cells. Sometimes the bodies were lying among masses of epithelial cells, but were not within epithelial cells. At times the spores had penetrated one-third of the way through the tumor.

Hence De-Meser reviews the literature upon the power of absorption of granulating surfaces. This review showed that the power of absorption possessed by granulating surfaces varies with the condition of the granulating surface and with the material placed upon it. Under certain circumstances soluble substances (*e. g.*, alkaloids) or insoluble substances (cinnabar) or living organisms (bacteria) can be absorbed.

The lycopodium spores were too large to enter through the lymphatic spaces, and De-Meser believes that the spores were caught in the furrows of the granulating surface and then were overgrown by the cancer.

The case is important because numerous observers have found blastomycetes in cancerous tissue and have believed that these bodies produced the epithelial growth. But since it is proved that lycopodium spores can enter cancerous tissue it is certain that blastomycetes can enter in the same way, and may be present accidentally and have nothing to do with the causation of the disease. Moreover, a number of men have inoculated animals with blastomycetes and produced nothing but granulomata; moreover, blastomycetes in cancers are few in number and have no regular relation to the cancerous process. Nor is the fact that blastomycetes are seen in the protoplasm of malignant tumors any argument that they cause proliferation of the epithelial cells, for blastomycetes have a limited power of motion, and in sarcomata the cells are of mesoblastic type, and hence may be phagocytic.—E. H. N.

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